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DATE: Tuesday, November 16, 2004

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☐	L4	cd11b same (189 or 316 or 332)	12
☐	L3	xiong-jian-ping.in.	6
☐	L2	li-rui.in.	13
☐	L1	arnaout-$-amin.in.	15

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     (FILE 'HOME' ENTERED AT 09:31:07 ON 16 NOV 2004)

     FILE 'STNGUIDE' ENTERED AT 09:32:13 ON 16 NOV 2004

     FILE 'DISSABS, 1MOBILITY, AGRICOLA, AQUASCI, BIOTECHNO, COMPENDEX,
     COMPUAB, CONF, CONFSCI, ELCOM, HEALSAFE, IMSDRUGCONF, LIFESCI, MEDICONF,
     OCEAN, PAPERCHEM2, PASCAL, POLLUAB, SOLIDSTATE, ADISCTI, ADISINSIGHT,
     ADISNEWS, ANABSTR, ANTE, AQUALINE, BIOBUSINESS, ...' ENTERED AT 09:32:27
     ON 16 NOV 2004
                E ARNAOUT M AMIN?/AU
L1          396 S E1 OR E2 OR E10
                E LI RUI?/AU
L2            2 S E1 OR E2
                E XIONG JIAN-PING?/AU
                E XIONG 'JIAN-PING'?/AU
                E XIONG JIANPING?/AU
L3           21 S E1 OR E2
L4           41 S CD11B (S) ILE
L5           19 S L4 (S) ( OPEN OR CLOSE OR ACTIVE OR NEO? OR CONFORMATION)
L6           14 DUP REM L5 (5 DUPLICATES REMOVED)
L7            8 S L4 (S) (316 OR 189)

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L6 ANSWER 8 OF 14 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 2000:943978 SCISEARCH
THE GENUINE ARTICLE: 381AH
TITLE: An isoleucine-based allosteric switch controls affinity and shape shifting in integrin CD11b A-domain
AUTHOR: Xiong J P; Li R; Essafi M; Stehle T; Arnaout M A (Reprint)
CORPORATE SOURCE: MASSACHUSETTS GEN HOSP, RENAL UNIT, LEUKOCYTE BIOL & INFLAMMAT PROGRAM, 149 13TH ST, CHARLESTOWN, MA 02129 (Reprint); MASSACHUSETTS GEN HOSP, RENAL UNIT, LEUKOCYTE BIOL & INFLAMMAT PROGRAM, CHARLESTOWN, MA 02129; HARVARD UNIV, SCH MED, CHARLESTOWN, MA 02129
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (8 DEC 2000) Vol. 275, No. 49, pp. 38762-38767.
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
ISSN: 0021-9258.
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LANGUAGE: English
REFERENCE COUNT: 37

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB In response to cell activation signals, integrins switch from a low to a high affinity state. Physiologic ligands bind to integrins through a von Willebrand Factor A-type domain. Crystallographic studies revealed two **conformations** of this domain, ''closed'' and ''**open**.'' The latter crystallizes in complex with a pseudoligand or ligand, suggesting that it represents the high affinity state; data linking structure and activity are lacking however. In this communication, we expressed stable low and high affinity forms of integrin **CD11b** A-domain and determined their binding isotherms and crystal structures. The low affinity form, generated by deleting an N-terminal extension extrinsic to the domain, did not bind to physiologic ligands, and crystallized in the closed **conformation**. The high affinity form was generated by either deleting or substituting an invariable C-terminal **Ile**(316), wedged into a hydrophobic socket in the closed form, but displaced from it in the **open** structure. Both mutants crystallized in the **open conformation**, and the Ile316 --> Gly-modified integrin displayed high affinity. Structural differences between the low and high affinity forms were detected in solution. These data establish the structure-function correlates for the **CD11b** A-domain, and define a ligand-independent isoleucine-based allosteric switch intrinsic to this domain that controls its **conformation** and affinity.